

Xerophthalmia in an Adult: A Case Report

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Abstract

Case Report

Vitamin A is essential for normal retinal function and it plays an important role in corneal and conjunctival epithelial cell. Vitamin A deficiency has been known to occur as a result of poor dietary intake, chronic liver diseases and gastrointestinal malabsorption. Here we report 30 years old male with history of chronic diarrhea and fatty liver presenting as bilateral corneal ulcer with Bitot spots and conjunctival xerosis with decreased macular thickness on optical coherence tomography (OCT)

Keywords: Corneal ulcer, Vitamin A deficiency, Xerophthalmia, Xerosis.

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INTRODUCTION

Vitamin A is a fat-soluble vitamin required for normal cellular growth and differentiation and has an important role in visual process. Vitamin A is essential for normal retinal function where it is vital for the synthesis of visual pigments in both rods and cones during photo transduction and it plays an important role in corneal and conjunctival epithelial cell ribonucleic acid (RNA) and glycoprotein synthesis. Vitamin A deficiency has been known to occur as a result of poor dietary intake, chronic liver diseases and gastrointestinal malabsorption [1-4]. Here we report 30 years old male with history of chronic diarrhea and vomiting and fatty liver presenting as bilateral corneal ulcer with Bitot spots and conjunctival xerosis with decreased macular thickness on optical coherence tomography (OCT).

CASE REPORT

A 30 year old male was referred to eye out patient department from medicine department with bilateral corneal ulceration of 8 weeks duration. Patient presented with history of chronic diarrhea, chronic vomiting and fatty liver with leucocytosis and acute red eyes. Patient was not immunocompromised. There was history of watering, redness in both eyes and he was complaining of night blindness. On ocular examination her best corrected visual acuity in right eye was 6/60 and in left eye was hand movement. On slit lamp examination there was corneal ulcer of size 2mm x1.5mm in infra-nasal quadrant close to limbus with surrounding stromal infiltration in the right eye and

there were multiple small sized corneal ulcers ranging from 1.5 to 2mm with surrounding stromal infiltration and Descemet's folds in the left eye. Bitot spots were present in both eyes in temporal conjunctiva.



Fig-1: Shows corneal haze and bitot spot

Intra ocular pressure was 14.6 and 17.3 mmHg in right and left eye respectively measured with Schiottz tonometer. Patient was diagnosed as case of Xerophthalmia stage III. On dilated fundus examination, fine peripheral pigmentary granularity was seen in the right eye.

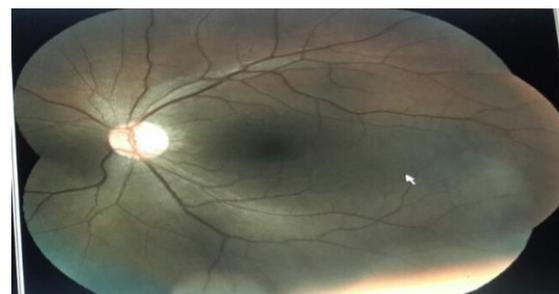


Fig-2: Shows pigmentary granularity in fundus

Fundus examination in left eye was not possible. OCT with Optovue was done and Retina map showed decreased macular thickness in right eye.

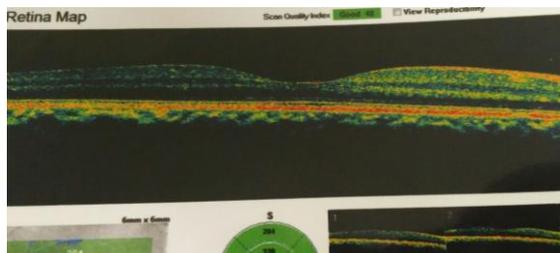


Fig-3: OCT shows decreased macular thickness in right eye.

OCT was not possible in left eye due to corneal haze. Patient was treated with topical moxifloxacin 0.5% one drp onehourly with cyclopentolate 1% twice daily. Therapeutic Vitamin A was also given with 2 lakh IU of Vitamin A suspension which was repeated after 24 hours and after 14 days. The ulcers healed within 2-3 weeks in both eyes. There was also improvement in the night vision.

DISCUSSION

Vitamin A deficiency has largely disappeared from the developed countries, but it still remains a prevalent problem in the developing countries. Vitamin A deficiency is caused by chronic malnutrition, variety of pathologies such as Celiac disease, biliary obstruction, cystic fibrosis, chronic liver diseases including alcoholic cirrhosis, autoimmune hepatitis and cirrhosis, hepatitis B or C etc [6-9]. An estimated 10 million preschool age children and pregnant women develop potentially blinding xerophthalmia each year [1]. Retinol and retinyl esters are the most common forms of preformed vitamin A in the human diet [5]. The natural dietary sources of vitamin a are animal tissues that are rich in retinyl esters, such as liver and green leafy vegetables which contains precursors of for carotene. These water insoluble vitamins are first hydrolyzed in the intestinal lumen to free retinol. Once it is absorbed the vitamin A is incorporated into chylomicrons. The chylomicrons are transported through the lymph and the general circulation, and are partially metabolized outside the liver. The chylomicron remnants, enriched in cholesterol and retinyl esters enter the liver, which is the main storage place for vitamin A in the body. In severe liver disease there is reduced production of retinol binding protein, reduced amount of zinc and reduced storage of vitamin A esters in the liver [4]. Ocular manifestations of Vitamin A deficiency include cornea and conjunctival xerosis, keratinization of the conjunctiva, keratomalacia, retinopathy, visual loss and nyctalopia [9]. The earliest ocular symptom of vitamin A deficiency is nyctalopia secondary to depletion of photo pigments. The storage form is retinol esterifies to fatty acids predominantly as retinyl palmitate. Retinal is the form involved in vision, where as retinoic acid is involved in growth and cellular

functions. Night blindness results when the vitamin A pool in the eye becomes depleted and the concentration in the rod cells is lowered [5]. Gopse *et al.* reported vitamin A deficiency due to panic-disorder related malnutrition [10]. McLaughlin *et al.* [11] reported a series of xerophthalmia due to alcoholic liver disease predicting a potential epidemic at our door step.

CONCLUSION

Vitamin A deficiency should be suspected in the presence of corneal ulceration with history of chronic diarrhea and liver disease which leads to malnutrition syndromes.

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